See petition to correct inventorship, received 9/27/04.
SLW, 11/30/04

Docket No

COMBINED DECLARATION FOR PATENT APPLICATION AND POWER OF ATTORNEY

As a below named inventor, I hereby declare that:

My residence, post office address and citizenship are as stated below next to my name,

I believe I am the original first and sole inventor (if only one name is listed below) or an original, first and joint inv names are listed below) of the subject matter which is claimed and for which a patent is sought on the inventi

SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE S

the specification of which (check one) X is attached hereto or _ was filed on _ as Application Serial No. _ and was ar applicable).

I hereby state that I have reviewed and understand the contents of the above-identified specification, including amended by any amendment referred to above.

I acknowledge the duty to disclose information which is material to the examination of this application in accordanc Code of Federal Regulations, Section 1.56.

I hereby claim foreign priority benefits under Title 35, United States Code, §119(a)-(d) of any foreign application(s inventor's certificate listed below and have also identified below any foreign application for patent or inventor's cer filing date before that of the application on which priority is claimed:

Prior Foreign Application(s): Prior Yes

Number	Country	Day/Month/Year Filed

I hereby claim the benefit under Title 35, United States Code, §119(e) of any United States provisional applications(s

Please see attached Appendix A (Listing 87 U.S. Provisional Patent Applications)

Application Ser. No. Filing Date

I hereby claim the benefit under Title 35, United States Code, §120 of any United States applications(s) listed belo as the subject matter of each of the claims of this application is not disclosed in the prior United States application provided by the first paragraph of Title 35, United States Code, §112, I acknowledge the duty to disclose material i defined in Title 37, Code of Federal Regulations, §1.56 which occurred between the filing date of the prior applic national or PCT international filing date of this application:

Please see attached Appendix B (Listing 20 U.S. applications and 27 PCT applications

Application Ser. No. Filing Date Status: Patented, Pending, Abandon

Application Ser. No. Filing Date Status: Patented, Pending, Abandon

POWER OF ATTORNEY: As a named inventor, I hereby appoint all Attorney(s) and/or Agent(s) associated with Patent Office Issued Customer Number to prosecute this application and transact all business in the Patent and Tra connected therewith.

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PATENT TRADEMARK OFFICE

Paul E. Rauch, Registration No. 38,591, CUSTOMER NUMBER: 28,442

quency of c-*erb*B-2 protein overproduction in comedocarcinoma in situ, compared with infiltrating ductal carcinoma, could be explained by the fact that many infiltrating ductal carcinomas arise from other types of intraductal carcinoma, which show c-*erb*B-2 activation infrequently. Others have speculated that carcinoma in situ with c-*erb*B-2 activation tends to regress or to lose c-*erb*B-2 activation during progression to invasion.[40,68,92] Infiltrating and in situ components of ductal carcinoma, however, usually are similar with respect to c-*erb*B-2 activation,[11,39] although some authors have noted more heterogeneity of the immunohistochemical staining pattern in invasive than in in situ carcinoma.[40,42,68] Activation of c-*erb*B-2 is infrequent in lobular carcinoma in situ. If lesions contain more than one histological pattern of carcinoma in situ, the c-*erb*B-2 protein overproduction tends to occur in the comedocarcinoma in situ but may include other areas of carcinoma in situ.[42,54,68] Overproduction of c-*erb*B-2 protein in ductal carcinoma in situ correlates with larger cell size and a periductal lymphoid infiltrate.[68]

Activation of c-*erb*B-2 has not been identified in benign breast lesions, including fibrocystic disease, fibroadenomas, and radial scars (Table 2). Strong membrane immunohistochemical reactivity for c-*erb*B-2 has not been described in atypical ductal hyperplasia, although weak accentuation of membrane staining has been noted infrequently.[39,42,54] In normal breast tissue, c-*erb*B-2 DNA is diploid, and c-*erb*B-2 is expressed at lower levels than in activated tumors.[34,35,65,68]

These preliminary data suggest that c-*erb*B-2 activation may not be useful for resolving many of the common problems in diagnostic surgical pathology. For example, c-*erb*B-2 activation is infrequent in tubular carcinoma and radial scars. In addition, because c-*erb*B-2 activation is unusual in atypical ductal hyperplasia, cribriform carcinoma in situ, and papillary carcinoma in situ, detection of c-*erb*B-2 activation in these lesions may not be helpful in their differential diagnosis. The histological features of comedocarcinoma in situ, which commonly overproduces c-*erb*B-2, are unlikely to be mistaken for those of benign lesions. Activation of

TABLE 2. c-*erb*B-2 ACTIVATION IN BENIGN HUMAN BREAST LESIONS

Histological Diagnosis	c-*erb*B-2 DNA Amplification[a]	c-*erb*B-2 mRNA Overproduction	c-*erb*B-2 Protein Overproduction
Fibrocystic disease	0/10[93]	—	0/32,[39] 0/9,[68] 0/8[65]
Atypical ductal hyperplasia	—	—	2(weak)/21,[54] 1(cytoplasmic)/13[39]
Benign ductal hyperplasia	—	—	0/12[39]
Sclerosing adenosis	—	—	0/4[39]
Fibroadenomas	0/16,[34] 0/6,[93] 0/2,[21] 0/1[91]	0/6,[35] 0/3[34]	0/21,[39] 0/10,[68] 0/8,[65] 0/3[42]
Radial scars	—	—	0/22[39]
Blunt duct adenosis	—	—	0/14[39]
"Breast mastosis"	—	0/3[35]	—

[a]Shown as number of cases with activation/number of cases studied; reference is given as a superscript.